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 NEWS  4  AUG 28   ADISCTI Reloaded and Enhanced
 NEWS  5  AUG 30   CA(SM)/CAplus(SM) Austrian patent law changes
 NEWS  6  SEP 11   CA/CAplus enhanced with more pre-1907 records
 NEWS  7  SEP 21   CA/CAplus fields enhanced with simultaneous left and right
                   truncation
 NEWS  8  SEP 25   CA(SM)/CAplus(SM) display of CA Lexicon enhanced
 NEWS  9  SEP 25   CAS REGISTRY(SM) no longer includes Concord 3D coordinates
 NEWS 10  SEP 25   CAS REGISTRY(SM) updated with amino acid codes for pyrrolysine
 NEWS 11  SEP 28   CEABA-VTB classification code fields reloaded with new
                   classification scheme
 NEWS 12  OCT 19   LOGOFF HOLD duration extended to 120 minutes
 NEWS 13  OCT 19   E-mail format enhanced
 NEWS 14  OCT 23   Option to turn off MARPAT highlighting enhancements available
 NEWS 15  OCT 23   CAS Registry Number crossover limit increased to 300,000 in
                   multiple databases
 NEWS 16  OCT 23   The Derwent World Patents Index suite of databases on STN
                   has been enhanced and reloaded
 NEWS 17  OCT 30   CHEMLIST enhanced with new search and display field
 NEWS 18  NOV 03   JAPIO enhanced with IPC 8 features and functionality
 NEWS 19  NOV 10   CA/CAplus F-Term thesaurus enhanced
 NEWS 20  NOV 10   STN Express with Discover! free maintenance release Version
                   8.01c now available
 NEWS 21  NOV 13   CA/CAplus pre-1967 chemical substance index entries enhanced
                   with preparation role

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               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

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=> oncolytic
L1         2297 ONCOLYTIC

=> oncolysis
L2          529 ONCOLYSIS

=> diagnosis
L3       907108 DIAGNOSIS

=> L1 and L3
L4           40 L1 AND L3

=> L1 and L2
L5          314 L1 AND L2

=> cancer and L4
L6           18 CANCER AND L4

=> cancer L5
MISSING OPERATOR CANCER L5
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> cancer and L5
L7          202 CANCER AND L5

=> phenotyp3
 75% OF LIMIT FOR TOTAL ANSWERS REACHED
L8      9482317 3

=> L8 and Cancel
L9         1780 L8 AND CANCEL

=> L8 and cancer
L10      222964 L8 AND CANCER

=> L10 and L1
L11         260 L10 AND L1

=> L10 and L3
L12       27130 L10 AND L3

=> diagnosis and L11
L13           7 DIAGNOSIS AND L11

=> diagnosis and L12
L14       27130 DIAGNOSIS AND L12

=> L12 and L2
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L15 1 L12 AND L2

=> D L13 IBIB ABS 1-7

L13 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:733735 CAPLUS
DOCUMENT NUMBER: 145:165508
TITLE: Nucleic acids encoding tumor suppressor, proapoptotic protein, cytokine, growth factor, hormone, tumor antigen or enzyme for diagnosis and therapy of cancer or hyperproliferative disease
INVENTOR(S): Clarke, Peter; Chada, Sunil; Menander, Kerstin; Sobol, Robert; Zhang, Shuyuan
PATENT ASSIGNEE(S): Introgen Therapeutics, Inc., USA
SOURCE: PCT Int. Appl., 164 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006079014	A2	20060727	WO 2006-US2255	20060120

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2005-645826P P 20050121
US 2005-692481P P 20050621

AB Compns. and methods for preventing or inhibiting the growth of a hyperproliferative lesion in a subject that include a nucleic acid comprised in a solid or semi-solid formation or in a transdermal or transcutaneous delivery device are disclosed. Also disclosed are compns. of a nucleic acid capable of preventing or inhibiting the growth of a hyperproliferative lesion in a subject that include an adhesive. Compns. of a nucleic acid capable of preventing or inhibiting the growth of a hyperproliferative lesion in a subject that include a nucleic acid uptake enhancer are also disclosed. Methods of preventing or inhibiting the growth of a hyperproliferative lesion in a subject that involve these therapeutic compns. and devices are also disclosed.

L13 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:20988 CAPLUS
DOCUMENT NUMBER: 140:73576
TITLE: Oncolytic viruses as phenotyping agents for neoplasms and use for tumor diagnosis and therapy
INVENTOR(S): Thompson, Bradley G.; Coffey, Matthew C.
PATENT ASSIGNEE(S): Oncolytics Biotech, Inc., Can.
SOURCE: PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2004003562	A2	20040108	WO 2003-CA951		20030625
WO 2004003562	A3	20040506			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
US 2004029112	A1	20040212	US 2003-602024		20030624
CA 2487824	AA	20040108	CA 2003-2487824		20030625
AU 2003245760	A1	20040119	AU 2003-245760		20030625
EP 1520175	A2	20050406	EP 2003-737795		20030625
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK					
BR 2003011983	A	20050426	BR 2003-11983		20030625
CN 1666105	A	20050907	CN 2003-815353		20030625
JP 2005531306	T2	20051020	JP 2004-516379		20030625
PRIORITY APPLN. INFO.:			US 2002-392031P	P	20020628
			US 2003-443188P	P	20030129
			WO 2003-CA951	W	20030625

AB The present invention provides a method of diagnosing neoplasms having a particular phenotype by using oncolytic viruses that selectively replicate in neoplasms having the particular phenotype. For example, reovirus does not replicate in normal cells. However, reovirus selectively replicate in cells with an activated ras pathway, which leads to death of these cells. Therefore, a cell which becomes neoplastic due to, at least in part, elevated ras pathway activities can be diagnosed by its susceptibility to reovirus replication. This invention can further be applied, using other oncolytic viruses, to the diagnosis and/or treatment of other tumors, such as interferon-sensitive tumors, p53-deficient tumors and Rb-deficient tumors. Kits useful in the diagnosis or treatment disclosed herein are also provided.

L13 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:716031 CAPLUS
DOCUMENT NUMBER: 137:242151
TITLE: Oncolytic RNA replicons
INVENTOR(S): Ansardi, David C.; Morrow, Casey D.; Porter, Donna C.
PATENT ASSIGNEE(S): University of Alabama Research Foundation, USA; Replicon Technologies, Inc.
SOURCE: PCT Int. Appl., 67 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002072027	A2	20020919	WO 2002-US7646	20020313
WO 2002072027	A3	20030918		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW				

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002306709	A1	20020924	AU 2002-306709	20020313
US 2003040498	A1	20030227	US 2002-97058	20020313
PRIORITY APPLN. INFO.:			US 2001-275840P	P 20010314
			WO 2002-US7646	W 20020313

AB The limited efficacy and/or toxicity of conventional therapies for many types of human cancers underscores the need for development of safe and effective alternative treatments. Towards this goal, the invention describes the direct oncolytic activity of RNA-based vectors derived from poliovirus, termed replicons, which are genetically incapable of producing infectious virus. Replicons of the invention are cytopathic in vivo for human tumor cells originating from brain, breast, lung, ovaries and skin (melanoma). Injection of replicons into established xenograft flank tumors in scid mice resulted in oncolytic activity and extended survival. Inoculation of replicons into established intracranial xenografts tumors in scid mice resulted in tumor infection and extended survival. Histol. anal. revealed that replicons infected tumors cells at the site of inoculation and, most importantly, diffused to infect tumor cells which had metastasized from the initial site of implementation. The wide spectrum of cytopathic activity for human tumors combined with effective distribution following in vivo inoculation establishes the therapeutic potential of poliovirus replicons for a variety of cancers. Replicons of the invention may addnl. comprise a heterologous nucleic acid with a min. length of one nucleotide. According to the invention, a heterologous nucleic acid is any nucleic acid that is not present in the genome of wildtype poliovirus. Thus, the invention contemplates a replicon having a transgene, a site-specific mutation (e.g. deletion, insertion, or substitution), a restriction site, a site-specific recombination site (e.g. loxP, FRT, and RS), an expression control sequence, or combinations thereof. Transgenes may confer or enhance oncolytic activity by various means. A transgene of the invention may also encode markers such as luciferase, an autofluorescent protein (e.g. green fluorescence protein), and 3 -glucuronidase. A transgene for use in the invention may also encode an immunogen.

L13 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER:	2001:659029 CAPLUS
DOCUMENT NUMBER:	136:48099
TITLE:	A hepatocellular carcinoma-specific adenovirus variant, CV890, eliminates distant human liver tumors in combination with doxorubicin
AUTHOR(S):	Li, Yuanhao; Yu, De-Chao; Chen, Yu; Amin, Pinky; Zhang, Hong; Nguyen, Natalie; Henderson, Daniel R.
CORPORATE SOURCE:	Calydon, Inc., Sunnyvale, CA, 94089, USA
SOURCE:	Cancer Research (2001), 61(17), 6428-6436 CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER:	American Association for Cancer Research
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB Hepatocellular carcinoma (HCC) is the third leading cause of cancer death in the world. Tumor resection remains the only curative treatment but is often not possible because of advanced stage and frequently unsuccessful because of intrahepatic or distant tumor recurrence. α-Fetoprotein (AFP), a tumor marker currently used for the diagnosis and management of HCC, is an oncofetal protein expressed in a majority of HCCs but rarely in normal hepatocytes. Because AFP gene expression is tightly regulated at the level of transcription, AFP transcriptional regulatory elements (TRE) are excellent candidates for generating HCC-specific oncolytic adenoviruses. We devised a new strategy for the AFP TRE to control an artificial E1A-IRES-E1B

bicistronic cassette in an adenovirus 5 vector (Ad5) and constructed an HCC-specific oncolytic virus, CV890. In vitro, CV890 expression of the E1A and E1B genes, virus replication, and cytopathic effects were examined by Northern blot, Western blot, virus yield assay, and 3 -(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide assay in AFP-producing cell lines (HepG2, Huh7, Hep3B, PLC/PRF/5, and SNU449), non-AFP-producing cell lines (Sk-Hep-1, Chang liver cell, LNCaP, HBL-100, PA-1, UM-UC-3, SW 780, Colo 201, and U118 MG), and non-AFP-producing human primary cells (lung fibroblast, bladder smooth muscle, and mammary epithelial). CV890 efficiently replicates in and destroys AFP-producing HCC cells as well as wild-type Ad5, but replication is highly attenuated in non-AFP-producing HCC cells or non-HCC cells. CV890 produced 5,000-100,000-fold less virus than wild-type Ad5 in non-AFP-producing cells. CV890 was attenuated 100-fold more than CV732, a virus containing the AFP TRE driving the E1A gene alone, in non-AFP-producing cells. These studies demonstrated that expression of both E1A and E1B genes under the control of a bicistronic AFP-E1A-IRES-E1B cassette yielded improvements in virus specificity equivalent to driving the E1A and E1B genes with two independent TREs yet requires only one TRE thereby conserving genomic space within the virus. Significantly, CV890 produced nearly the same yield of virus in cells that produced AFP over a 75-fold range, from a low of 60 ng AFP/106 cells/10 days to as high as 4585 ng AFP/106 cells/10 days. In vivo, antitumor efficacy of CV890 was examined in BALB/c-nu/nu mice containing large s.c. HepG2 or Hep3B tumor xenografts. Tumor volume of distant xenografts dropped below baseline 4 wk after a single i.v. injection. Combination of CV890 with doxorubicin demonstrated synergistic antitumor efficacy, yielding complete elimination of distant Hep3B tumors 4 wk after a single i.v. administration of both compds. Our results support the clin. development of CV890 as an antineoplastic agent for the treatment of localized or metastatic HCC.

REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 5 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:586592 BIOSIS

DOCUMENT NUMBER: PREV200300570046

TITLE: Phase I study of replication-competent adenovirus-mediated double-suicide gene therapy in combination with conventional-dose three-dimensional conformal radiation therapy for the treatment of newly diagnosed, intermediate- to high-risk prostate cancer.

AUTHOR(S): Freytag, Svend O. [Reprint Author]; Stricker, Hans; Pegg, Jan; Paielli, Dell; Pradhan, Deepak G.; Peabody, James; Deperalta-Venturina, Mariza; Xia, Xueqing; Brown, Steve; Lu, Mei; Kim, Jae Ho

CORPORATE SOURCE: Molecular Biology Research, Henry Ford Health System, One Ford Place, Wing 5D, Detroit, MI, 48202-3450, USA sfreytal@hfhs.org

SOURCE: Cancer Research, (November 1 2003) Vol. 63, No. 21, pp. 7497-7506. print.
ISSN: 0008-5472 (ISSN print).

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 10 Dec 2003
Last Updated on STN: 10 Dec 2003

AB The primary study objective was to determine the safety of intraprostatic administration of a replication-competent, oncolytic adenovirus containing a cytosine deaminase (CD)/herpes simplex virus thymidine kinase (HSV-1 TK) fusion gene concomitant with increasing durations of 5-fluorocytosine and valganciclovir prodrug therapy and conventional-dose three-dimensional conformal radiation therapy (3D-CRT) in patients with newly diagnosed, intermediate- to high-risk prostate cancer. Secondary objectives were to determine the persistence of therapeutic transgene expression in the prostate and to examine early posttreatment

response. Fifteen patients in five cohorts received a single intraprostatic injection of 1012 viral particles of the replication-competent Ad5-CD/TKrep adenovirus on day 1. Two days later, patients were administered 5-fluorocytosine and valganciclovir prodrug therapy for 1 (cohorts 1-3), 2 (cohort 4), or 3 (cohort 5) weeks along with 70-74 Gy 3D-CRT. Sextant needle biopsy of the prostate was obtained at 2 (cohort 1), 3 (cohort 2), and 4 (cohort 3) weeks for determination of the persistence of transgene expression. There were no dose-limiting toxicities and no significant treatment-related adverse events. Ninety-four percent of the adverse events observed were mild to moderate and self-limiting. Acute urinary and gastrointestinal toxicities were similar to those expected for conventional-dose 3D-CRT. Therapeutic transgene expression was found to persist in the prostate for up to 3 weeks after the adenovirus injection. As expected for patients receiving definitive radiation therapy, all patients experienced significant declines in prostate-specific antigen (PSA). The mean PSA half-life in patients administered more than 1 week of prodrug therapy was significantly shorter than that of patients receiving prodrugs for only 1 week (0.6 versus 2.0 months; $P < 0.02$) and markedly shorter than that reported previously for patients treated with conventional-dose 3D-CRT alone (2.4 months). With a median follow-up of only 9 months, 5 of 10 (50%) patients not treated with androgen-deprivation therapy achieved a serum PSA ltoreq 0.5 ng/ml. The results demonstrate that replication-competent adenovirus-mediated double-suicide gene therapy can be combined safely with conventional-dose 3D-CRT in patients with intermediate- to high-risk prostate cancer . The shorter than expected PSA half-life in patients receiving more than 1 week of prodrug therapy may suggest a possible interaction between the oncolytic adenovirus and/or double-suicide gene therapies and radiation therapy.

L13 ANSWER 6 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:451747 BIOSIS
DOCUMENT NUMBER: PREV200100451747
TITLE: A hepatocellular carcinoma-specific adenovirus variant, CV890, eliminates distant human liver tumors in combination with doxorubicin.
AUTHOR(S): Li, Yuanhao; Yu, De-Chao; Chen, Yu; Amin, Pinky; Zhang, Hong; Nguyen, Natalie; Henderson, Daniel R. [Reprint author]
CORPORATE SOURCE: Calydon, Inc., 1324 Chesapeake Terrace, Sunnyvale, CA, 94089, USA
dhenderson@calydon.com
SOURCE: Cancer Research, (September 1, 2001) Vol. 61, No. 17, pp. 6428-6436. print.
CODEN: CNREA8. ISSN: 0008-5472.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 19 Sep 2001
Last Updated on STN: 22 Feb 2002

AB Hepatocellular carcinoma (HCC) is the third leading cause of cancer death in the world. Tumor resection remains the only curative treatment but is often not possible because of advanced stage and frequently unsuccessful because of intrahepatic or distant tumor recurrence. alpha-Fetoprotein (AFP), a tumor marker currently used for the diagnosis and management of HCC, is an oncofetal protein expressed in a majority of HCCs but rarely in normal hepatocytes. Because AFP gene expression is tightly regulated at the level of transcription, AFP transcriptional regulatory elements (TRE) are excellent candidates for generating HCC-specific oncolytic adenoviruses. We devised a new strategy for the AFP TRE to control an artificial E1A-IRES-E1B bicistronic cassette in an adenovirus 5 vector (Ad5) and constructed an HCC-specific oncolytic virus, CV890. In vitro, CV890 expression of the E1A and E1B genes, virus replication, and cytopathic effects were

examined by Northern blot, Western blot, virus yield assay, and 3 -(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide assay in AFP-producing cell lines (HepG2, Huh7, Hep3B, PLC/PRF/5, and SNU449), non-AFP-producing cell lines (Sk-Hep-1, Chang liver cell, LNCaP, HBL-100, PA-1, UM-UC-3, SW 780, Colo 201, and U118 MG), and non-AFP-producing human primary cells (lung fibroblast, bladder smooth muscle, and mammary epithelial). CV890 efficiently replicates in and destroys AFP-producing HCC cells as well as wild-type Ad5, but replication is highly attenuated in non-AFP-producing HCC cells or non-HCC cells. CV890 produced 5,000-100,000-fold less virus than wild-type Ad5 in non-AFP-producing cells. CV890 was attenuated 100-fold more than CV732, a virus containing the AFP TRE driving the E1A gene alone, in non-AFP-producing cells. These studies demonstrated that expression of both E1A and E1B genes under the control of a bicistronic AFP-E1A-IRES-E1B cassette yielded improvements in virus specificity equivalent to driving the E1A and E1B genes with two independent TREs yet requires only one TRE thereby conserving genomic space within the virus. Significantly, CV890 produced nearly the same yield of virus in cells that produced AFP over a 75-fold range, from a low of 60 ng AFP/106 cells/10 days to as high as 4585 ng AFP/106 cells/10 days. In vivo, antitumor efficacy of CV890 was examined in BALB/c-nu/nu mice containing large s.c. HepG2 or Hep3B tumor xenografts. Tumor volume of distant xenografts dropped below baseline 4 weeks after a single i.v. injection. Combination of CV890 with doxorubicin demonstrated synergistic antitumor efficacy, yielding complete elimination of distant Hep3B tumors 4 weeks after a single i.v. administration of both compounds. Our results support the clinical development of CV890 as an antineoplastic agent for the treatment of localized or metastatic HCC.

L13 ANSWER 7 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:512691 BIOSIS
DOCUMENT NUMBER: PREV200000512691
TITLE: Gene therapy for brain tumors: The fundamentals.
AUTHOR(S): Engelhard, Herbert H. [Reprint author]
CORPORATE SOURCE: Departments of Neurosurgery and Molecular Genetics, University of Illinois at Chicago, 912 South Wood St., Chicago, IL, 60612, USA
SOURCE: Surgical Neurology, (July, 2000) Vol. 54, No. 1, pp. 3-9. print.
ISSN: 0090-3019.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Nov 2000
Last Updated on STN: 11 Jan 2002

AB BACKGROUND: Over the past two decades, significant advances have been made in the fields of virology and molecular biology, and in understanding the genetic alterations present in brain tumors. The knowledge gained has been exploited for use in gene therapy. OBJECTIVE: The purpose of this article is to present an introduction to the field of brain tumor gene therapy for the practicing clinician. RESULTS: A variety of gene therapy strategies have now been used in the laboratory and in clinical trials for brain tumors. They can be divided into five categories: 1) gene-directed enzyme prodrug ("suicide gene") therapy (GDEPT); 2) gene therapy designed to boost the activity of the immune system against cancer cells; 3) oncolytic virus therapy; 4) transfer of potentially therapeutic genes-such as tumor suppressor genes-into cancer cells; and 5) antisense therapy. GDEPT is the strategy that has been most extensively studied. CONCLUSIONS: To date, gene therapy has been found to be reasonably safe and concerns related to adverse events such as insertional mutagenesis have not been realized. Although patients have not been cured, the development of this therapy could still be considered to be at an early stage. Current research is addressing factors that could be limiting the successful clinical application of gene therapy, which remains an intriguing experimental option for patients with

malignant brain tumors.

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=> Therapeutic adj diagnosis
L16           0 THERAPEUTIC ADJ DIAGNOSIS

=> therapeutic (w) diagnosis
L17         134 THERAPEUTIC (W) DIAGNOSIS

=> L1 and L17
L18           0 L1 AND L17

=> L2 andf L17
MISSING OPERATOR L2 ANDF
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> L2 and L17
L19           0 L2 AND L17
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